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Author: Dr Jessica McClelland Bethan Dalton Maria Kekic Savani Bartholdy Prof Iain C. Campbell Prof Ulrike Schmidt

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A systematic review of temporal discounting in eating disorders and obesity: behavioural and neuroimaging findings

Authors

Dr Jessica McClelland*[a] jessica.mcclelland@kcl.ac.uk

Ms Bethan Dalton [a] bethan.dalton@kcl.ac.uk

Ms Maria Kekic [a] maria.kekic@kcl.ac.uk

Ms Savani Bartholdy [a] savani.bartholdy@kcl.ac.uk

Prof Iain C. Campbell [a] iain.campbell@kcl.ac.uk

Prof Ulrike Schmidt [a] ulrike.schmidt@kcl.ac.uk

*Corresponding author: jessica.mcclelland@kcl.ac.uk (0044) 0207 848 0183

[a] Section of Eating Disorders, Department of Psychological Medicine, King's College London, Institute of Psychiatry, Psychology and Neuroscience, De Crespigny Park, London SE5 8AF, Fax: (0044) 0207 848 0182

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Highlights

- Temporal discounting (TD) tasks can be used to explore self-regulatory control.
- Limited data suggest increased TD in obesity, BED and BN, data in AN are mixed. .
- Frontostriatal neurocircuitry is implicated in TD and related to clinical outcomes.
- TD tasks are heterogeneous, limiting comparability and generalisability of results.
- TD may be multidimensional, state-related and important in treatment of ED/obesity.

ABSTRACT

Objective: Eating Disorders (ED) and obesity are suggested to involve a spectrum of self-regulatory control difficulties. Temporal discounting (TD) tasks have been used to explore this idea. This systematic review examines behavioural and neuroimaging TD data in ED and obesity.

Method: Using PRISMA guidelines, we reviewed relevant articles in MEDLINE, PsycINFO and Embase from inception until 17th August 2016. Studies that reported behavioural differences in TD and/or TD neuroimaging data in ED/obesity were included.

Results: Thirty-one studies were included. Limited data suggest that BN, BED and obesity are associated with increased TD, whilst data in AN are mixed. Aberrant neural activity in frontostriatal circuitry is implicated. TD tasks vary widely and TD in ED/obesity may vary according to factors such as illness stage.

Conclusion: Our findings suggest altered self-regulatory control in ED and obesity. TD tasks are heterogeneous, limiting generalisability of findings. Research into whether TD is

multidimensional, along with transdiagnostic neuroimaging research is needed. Assessment of TD may be useful in psychoeducation, outcome prediction and treatment of ED/obesity.

Keywords: eating disorders; obesity, temporal discounting, delay discounting, impulsivity, delayed gratification.

1. INTRODUCTION

1.1 Eating disorders and obesity

Eating disorders (ED) are serious mental illnesses which affect up to 3.5% of women and 2.0% of men (Schmidt et al., 2016). Anorexia nervosa (AN) has a restrictive (AN-R) and a binge/purge (AN-B/P) subtype: both involve severe food restriction, and AN-B/P also involves binge eating and/or purging behaviours (e.g. vomiting, laxative misuse). Bulimia nervosa (BN) and binge eating disorder (BED) are characterised by frequent binge eating and, whilst BN involves regular compensatory behaviours, BED does not (A.P.A, 2013). Mortality rates in ED are among the highest of all psychiatric conditions (Arcelus et al., 2011; Fichter et al., 2008).

Unlike ED, which are diagnosable psychiatric disorders, obesity is a wide-spread physical health condition. It is characterised by overeating leading to weight gain, and is often associated with reduced psychological well-being (Magallares et al., 2014). Globally, at least 13% of adults are clinically obese and worldwide prevalence rates have more than doubled over the past 40 years (BMI > 30kg/m^2; WHO, 2015). Raised BMI is a major risk factor for many non-fatal but disabling disorders (e.g. osteoarthritis), is associated with some of the leading causes of death (e.g. diabetes, cardiovascular disease and cancer; Kaiser, 2013; Lu et al., 2014; Yoshimoto et al., 2013) and a BMI indicative of obesity is thought to confer a 2- to 10-year decrease in life expectancy (Whitlock et al., 2009). Obesity is a growing and serious public health issue, it is a major economic and even environmental burden (Dannenberg et al., 2004; Wang et al., 2011).

It is important to emphasise that obesity and ED are distinct physical and psychiatric conditions, respectively. However, co-morbidity rates between ED and obesity (lifetime and familial) are high. For example, the prevalence of lifetime obesity in ED is reportedly 28%, ranging from 5% in AN to 87% in BED. Over the last 10 years there has been a threefold

increase in obesity in the ED, with prevalence rates predicted to continue to rise. ED patients with lifetime obesity display higher ED severity, worse general psychopathology and have a poorer prognosis than ED patients without lifetime obesity (Villarejo et al., 2012). There also seems to be a clear distinction between obesity and obesity with BED. The latter is associated with more severe obesity, greater medical and psychiatric comorbidity, greater functional impairment and poorer treatment outcomes (Bulik et al., 2002; Fandino et al., 2010; Hsu et al., 1998).

Obesity and ED have a number of overlapping biopsychosocial risk factors, e.g. (epi)genetics, personality traits, ethnicity, adverse events and neurobehavioural processes (Haines et al., 2010; Jacobi et al., 2004; Stunkard, 1988). Studies using a number of neurobehavioural tasks (e.g. Iowa Gambling Task, Wisconsin Card Sorting Test, Stroop, Go-No-Go, Stop Signal Task) demonstrate shared impairments in reward processing and executive functions across ED and obesity. Specifically, reviews and meta-analyses suggest that ED and obesity are associated with comparable neurocognitive difficulties across decision-making, planning, problem solving, cognitive flexibility, reward processing and response inhibition (Bartholdy et al., 2016; Fagundo et al., 2012; Lavagnino et al., 2016; Wu et al., 2014, 2016).

In ED, these difficulties are thought to be underpinned by aberrant frontostriatal neural circuitry, manifesting in impaired regulation of appetite, emotion and self-control (Friederich et al., 2013; Kaye et al., 2011; Kessler et al., 2016; Marsh et al., 2009a; Marsh et al., 2009b). More specifically, altered functioning of 'bottom-up' mesolimbic regions (e.g. amygdala, striatum) in conjunction with either reduced or exaggerated 'top-down' cognitive control (via the prefrontal cortex, PFC) are seen as contributing to impulsive (e.g. BN, BED) or exaggerated self-control (e.g. AN) related symptoms/behaviours in ED (Ehrlich et al.,

2015; Friederich et al., 2013; Hege et al., 2015; Kaye et al., 2009; Kessler et al., 2016; Marsh et al., 2009b; Sanders et al., 2015).

In people who are obese, impaired executive functions are thought to be underpinned by altered neural activity in areas involved in reward processing (e.g. striatum, insula), emotion and memory (e.g. amygdala), homeostatic regulation of food intake (e.g. hypothalamus) and cognitive control (e.g. PFC) (Carnell et al., 2012; Lavagnino et al., 2016; Pursey et al., 2014; Stice and Yokum, 2016; Tuulari et al., 2015). More specifically, heightened reward sensitivity to food cues (Pursey et al., 2014) in conjunction with reduced cognitive control mechanisms (Tuulari et al., 2015) are proposed to contribute to maladaptive impulse control behaviours in obesity, such as the overconsumption of food. Such processes, similar to those described in addictive behaviours, are likely to contribute to the inability to regulate appetite, food intake and the development of impulsive and often compulsive eating habits (Ziauddeen et al., 2015).

As such, ED, and to a lesser extent obesity, have been considered within spectrum models of self-regulatory control (Brooks et al., 2012; Kaye et al., 2010; Marsh et al., 2007; Piccinni et al., 2015). However, some data suggest that ED may not fit on to a simple continuum. For example, patients with BN are reported to be more impulsive than patients with BED across a number of domains, e.g., self-harm, substance misuse (Hudson et al., 2007). Secondly, whilst AN is hypothesised to be at the 'self-controlled' end of the spectrum, people with AN are reported to display impaired inhibitory control (Galimberti et al., 2012) and behavioural impulsivity (Butler and Montgomery, 2005; Claes et al., 2006). Furthermore, patients with AN report a lack of control over symptoms and compulsivity is proposed as a central component of the disorder (Godier and Park, 2014; Godier and Park, 2015). Thus, ED may not lie on a simple linear spectrum model, but rather may involve a number of overlapping self-regulatory control difficulties, including impulsivity, compulsivity and

behavioural inhibition. These difficulties are also pertinent to obesity, yet it is unclear if they too map clearly on to a spectrum of disordered eating (Brooks et al., 2012; Piccinni et al., 2015).

1.2 Temporal discounting

Such self-regulation difficulties are implicated in intertemporal choice behaviour – i.e. impulsivity (the degree to which an individual acts in a spontaneous, unplanned fashion), compulsivity (the tendency to make plans and stick with them) and inhibition (the ability to withhold automatic or inappropriate behavioural responses; Frederick et al., 2002). Intertemporal choice behaviours were first explored in the well-known Stanford marshmallow experiment (Mischel et al., 1972) in which children had to refrain from eating one marshmallow in order to receive two at a later stage. Current tasks, enhanced by neuroeconomic principles, are conceptually identical to this: they involve a number of binary choices between a lesser valued reward available immediately, i.e. 'smaller-sooner' (SS), and a reward of larger magnitude available after a delay, i.e. 'larger-later' (LL) (Estle et al., 2007; Odum and Rainaud, 2003). Based on the observation that the subjective value of a reward decreases as a function of its temporal delay (Green et al., 1999), the rate at which a future reward is devalued is referred to as *delay discounting (DD)* or *temporal discounting (TD*; the terms are interchangeable but herein, TD is used throughout). An inclination to choose the SS reward, and thus the rate at which a LL reward is devalued, is thought to reflect choice impulsivity and an inability to delay gratification (see Fig 1).

Whilst rates of discounting vary between individuals, TD behaviour is proposed to be a relatively stable and predictive personality trait. For example, the children from the Stanford marshmallow experiment (Mischel et al., 1972) who initially demonstrated an ability to delay gratification had greater academic and personal success in adulthood and a lower BMI up to three decades later (Mischel et al., 1989; Schlam et al., 2013; Shoda et al.,

1990). Given such validity, TD has been explored across a range of mental disorders. In most disorders considered to date, increased rates of TD have been reported: these include conduct disorder (White et al., 2014), autism (Chantiluke et al., 2014), attention-deficit/hyperactivity disorder (ADHD; Demurie et al., 2012), schizophrenia (Heerey et al., 2007), major depressive disorder (MDD; Pulcu et al., 2014) and also in individuals demonstrating addictive-type behaviours (Reynolds, 2006; Story et al., 2014) and high levels of social anxiety (Rounds et al., 2007). Such findings are argued to reflect increased impulsivity in such disorders and broadly, across psychiatric conditions more generally. Using an alternative approach, Story et al. (2015) incorporated motivations for discounting future rewards, the accumulation of previous experiences and neurobiological mechanisms, to explain increased rates of TD in psychiatric disorders. Moreover, as mentioned, Frederick et al. (2002) proposed that intertemporal choice is multidimensional and that it involves impulsive, as well as compulsive and inhibitory control mechanisms. Whilst TD has not been investigated across all psychiatric illnesses, the only disorders to date that have not been associated with increased rates of TD, and in fact show the opposite, are AN (Decker et al., 2015; Steinglass et al., 2012) and obsessive compulsive personality disorder (OCPD; Pinto et al., 2014). There is also evidence to suggest that altered rates of TD in some psychiatric conditions, e.g. AN (Decker et al., 2015; Wierenga et al., 2015) and MDD (Pulcu et al., 2014), could be state related.

The largest body of evidence for altered TD within a psychiatric condition is in relation to addictive disorders. This is of interest given the parallels often drawn between addictions and ED and obesity, in terms of shared patterns of addictive and compulsive behaviours and common underlying mechanisms. For example, important similarities between addictive disorders, ED and obesity include their presenting clinical features (e.g. cravings for food/drugs, overconsumption of food/drugs, inability to regulate food/drug

intake etc.), neuropsychological profiles (e.g. impulsivity, compulsivity, poor behavioural inhibition etc.) and shared neurobiological mechanisms (e.g. altered reward processing and cognitive control, neurotransmitter functioning and frontostriatal circuitry etc.) (Barry et al., 2009; Davis and Claridge, 1998; Gearhardt et al., 2011; Godier and Park, 2015; Kaye et al., 2013; O'Hara et al., 2015; Piccinni et al., 2015; Schulte et al., 2016; Smith and Robbins, 2013; Volkow and Baler, 2015; Ziauddeen et al., 2015). Given such similarities, the utility of TD in understanding addictive disorders (Amlung et al., 2016; Bickel et al., 2014a) is also informative for ED and obesity. Moreover, like addictive disorders, the concept of TD is highly relevant to the phenotype of ED and obesity – these conditions are typified by conflict between immediate gratification (e.g. drug-use/eating) and long-term goals (e.g. sobriety/weight loss or gain).

Research of TD in relation to addictive behaviour has shown that increased rates of TD predict initiation and/or future use of cigarettes and cocaine in high-school students (Audrain-McGovern et al., 2009; Ayduk et al., 2000) . Moreover, individuals who smoke or are dependent on opioids, cocaine or methamphetamine (Hofmeyr et al., 2016; Mejia-Cruz et al., 2016; Robles et al., 2011) display increased rates of TD and higher rates of discounting is proposed to be an index of dependence severity (Amlung et al., 2016). Following a variety of treatments (e.g. cognitive behaviour therapy, working memory training) for substance dependence (e.g. opioid, cocaine, smoking), reductions in rates of TD have been found (Bickel et al., 2011; Black and Rosen, 2011; Landes et al., 2012; Secades-Villa et al., 2014), which have often also been associated with a decrease in substance use (Black and Rosen, 2011; Yi et al., 2008), Finally, altered activity in brain areas involved in cognitive and emotional control (e.g. dorsolateral prefrontal cortex, DLPFC; anterior cingulate cortex, ACC; insula; amygdala) have been reported during TD tasks in methamphetamine users and people who abuse alcohol (Amlung et al., 2014; Claus et al., 2011; Hoffman et al., 2008).

Therefore, given the informative nature of TD in addictive disorders, exploring discounting behaviour in ED and obesity is of interest.

As has been shown in addictive disorders, neuroimaging research aids understanding of the neural underpinnings of TD and conditions associated with altered intertemporal choice behaviour. TD tasks are proposed to resemble everyday decision-making involving conflict between the pleasure seeking, emotional and impulsive 'hot' system and the contemplative, deliberate and rationale 'cool' system (Metcalfe and Mischel, 1999). Neuroimaging data are consistent with this; activity in mesolimbic areas, e.g. nucleus accumbens, ventral striatum and posterior cingulate cortex, has consistently been associated with the encoding of stimulus value and magnitude of reward (Ballard and Knutson, 2009; Kable and Glimcher, 2010; Li et al., 2013). Modulation of these reward valuation signals, along with temporal delay considerations are proposed to involve cortical regions, e.g. the DLPFC and posterior parietal cortex (Ballard and Knutson, 2009; Hare et al., 2014; Li et al., 2013; McClure et al., 2004). Moreover, the functional connectivity between these reward processing (e.g. striatum) and cognitive (e.g. DLPFC) brain regions is argued to be inversely correlated with TD behaviour (Hare et al., 2014; Li et al., 2013; McClure et al., 2004). In other words, a reduced exchange of information between these 'hot/emotive' and 'cool/cognitive' systems may contribute to increased TD and related psychopathologies. As mentioned, these neural pathways have also been implicated in the aetiology of ED and obesity (Kaye et al., 2010; Ziauddeen et al., 2015).

1.3 Aims

TD tasks are a novel way to explore self-regulatory control behaviour and its neural underpinnings. Despite a recent surge in literature on TD in ED and in obesity, along with associated neuroimaging data, there is yet, to be a formal review of this topic. Additionally, TD tasks differ widely, so methodological differences need to be considered to inform future

work. Therefore, the aim of this review was to explore TD behaviour and related neural activity in people with an ED or in individuals who are obese. More specifically, this review aims to address the following questions:

i) Do individuals with an ED or those that are obese display altered TD behaviour compared to healthy controls (HC)?

ii) Does TD behaviour differ according to the ED diagnoses and obesity?

iii) Is TD behaviour in people with an ED and/or obesity associated with specific/altered neural activity?

iv) How do TD tasks used in ED and obesity research differ and what are the implications of this?

2. METHODS

This review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009).

2.1 Selection criteria

Studies in English of any design that investigated TD in people with an ED and obesity were eligible for inclusion. Publications were included if they reported group differences in TD behaviour and/or the neural correlates of TD in people who have an ED or are obese. Studies were included regardless of whether or not their sample people with an ED/obesity also had co-morbid conditions (e.g. depression, substance abuse etc.).

Studies were excluded if they i) did not report group differences in behavioural TD *or* did not report neuroimaging data in people who were obese/had an ED, ii) investigated the effects of an intervention on TD behaviour or iii) used atypical TD tasks (i.e. probabilistic discounting, self-report questionnaires with < 2 binary choices). Review articles, meta-analyses, conference proceedings/abstracts, and unpublished theses were also not included.

2.2 Search strategy

Three electronic databases (MEDLINE, Embase, and PsycINFO) were searched (via OvidSP) from inception until 17th August 2016 using the following keywords, which were mapped to Medical Subject Headings where possible: temporal discounting, delay* discounting, discounting, reward discounting, intertemporal choice, intertemporal decision-making, delay* gratification and delay* reward in combination with eating disorder*, anorexi*, bulimi*, binge, obes*, overweight, weight, body, body mass index, BMI, diet, restrain*, and eat*. Searches were limited to English language and human participants. These searches were supplemented by internet searches and hand-searches of reference lists of potentially relevant papers and reviews. Citation tracking in Google Scholar was also performed.

Titles and abstracts of all retrieved publications were imported into EndNote, duplicates were removed, and papers that were deemed highly unlikely to be relevant were disregarded. Full-text versions of the remaining articles were then obtained and screened according to the pre-specified eligibility criteria. All papers that did not meet the inclusion criteria were excluded, with the reasons documented (Fig 2). The entire search process was conducted independently by two reviewers (M.K. and B.D.) and disagreements at the final stage were resolved via discussion with two additional reviewers (J.M. and S.B.).

2.3 Data extraction

Two reviewers (B.D. and S.B.) extracted data from all included studies into an electronic summary table, which was then checked by two other reviewers (J.M. and M.K.). Information collected related to sample size and characteristics, study design, task parameters (including reward type, framing of rewards, and measurement of TD) and relevant findings. A narrative synthesis is presented due to the methodological diversity of the included studies.

3. RESULTS

3.1 Characteristics of included studies

3.1.1 Participants. We identified 31 studies reported in 33 publications (including data from a total of 4546 participants) that met the inclusion criteria for this review. The majority (21 studies, *n*=3662) explored TD in individuals who were obese. The remaining studies compared HC to people with AN (4 studies, *n*=326, AN *n*=152), BN (1 study, *n*=96, BN *n*=39) or BED/loss-of-control eating (5 studies, *n*=543, BED/LOC *n*=174; all of which were overweight/obese individuals).

3.1.2 Primary outcome. Most studies reported behavioural differences in TD only (25 studies, *n*=4316; see Table 1), 3 reported TD-related neuroimaging data only (*n*=58; see Table 2) and 3 studies reported both behavioural and neuroimaging TD data (*n*=185;).

3.1.3 Methodological characteristics. Findings regarding the methodology of studies are presented in Table 3. This includes information regarding the TD task used, e.g. actuality of reward receipt (i.e. hypothetical or real-life), reward reinforcers (e.g. money, food etc.), administration (e.g. computerised, questionnaire etc.), reward framing (i.e. accelerate: SS reward variable/LL reward fixed; or delay: SS reward fixed/LL reward variable), calculation of TD (e.g. AUC, *k* etc.; see Table 4) and confounding variables (e.g. age, IQ, psychopathology etc.). Other methodological factors of interest are the type of delay period (months, weeks, days etc.), number of delay periods (ranging from 1 to 22) and number of binary choices, however these varied too widely to quantify coherently.

3.2 Behavioural findings (see Table 1)

3.2.1 Anorexia Nervosa (4 studies)

Findings regarding TD in people with AN are mixed. Initial data suggested that compared to controls, adult inpatients with current AN had reduced rates of TD: this enhanced ability to delay gratification was most pronounced in AN-R (Decker et al., 2015; Steinglass et al., 2012). Whilst Ritschel et al. (2015) did not find differences in TD between currently ill AN and HC, their participants were ~10 years younger (i.e. they included

adolescents in their AN sample), who had a much shorter illness duration than those in Steinglass et al. (2012) and Decker et al. (2015).

Ritschel et al. (2015) and others found that following weight restoration, remission and/or recovery from AN, TD rates were comparable to those in HC (Decker et al., 2015; Wierenga et al., 2015). However, in at least two of these studies (not reported in Wierenga et al., 2015) the weight-restored/recovered individuals still exhibited significantly higher ED psychopathology compared to HC (Decker et al., 2015; Ritschel et al., 2015). Therefore, the limited existing data, albeit mixed, suggest that reduced TD, i.e. an exaggerated ability to delay gratification, may be present only in currently ill adults with AN. Data suggest that this may be attributable to the state of undernutrition, rather than the psychological profiles of people with/recovered from AN.

3.2.2 Bulimia Nervosa (1 study)

To our knowledge, our recent study provides the only comprehensive data regarding TD in BN (Kekic et al., 2016). There are published conference abstract data, however, these were not eligible for inclusion in this review (Kaye et al., 2015; Wierenga et al., 2014). In our study, we found that, compared to HC, people with BN demonstrated increased rates of TD and thus choice impulsivity.

3.2.3 Binge Eating Disorder/Loss of Control (5 studies)

In the first study to examine TD in relation to BED, obese women with and without BED demonstrated comparable rates of TD. Both of these groups (i.e. obese and BED) displayed increased TD compared to HC. However, when differences in education were accounted for, these findings were no longer significant (Davis et al., 2010). The finding of increased TD in obese people (both with and without BED) compared to HC was replicated by Mole et al. (2015), who also demonstrated no differences in TD behaviour between the

obese with/without BED groups. The obese group (with and without BED) had comparable rates of TD to alcohol dependent (but abstinent) individuals.

However, across four different reward types (food, money, sedentary activity and massage time) obese people with BED demonstrated increased rates of TD in comparison to both obese people without BED and HC (Manwaring et al., 2011). These results (in relation to monetary rewards only) were recently replicated in overweight BED versus overweight control groups (Manasse et al., 2015a; Manasse et al., 2015b). Of note, the same group found no differences in TD behaviour in overweight/obese people with or without loss-of-control eating, i.e. subclinical symptomatology relevant to BED (Manasse et al., 2014). Of note, all BED studies reviewed here were in overweight/obese individuals. To our knowledge there are no data regarding TD in normal weight people with BED.

3.2.4 Obesity (18 studies)

Of the 18 studies reporting behavioural TD findings in obesity, only two studies specifically state that they excluded individuals with a current/past ED (Hendrickson and Rasmussen, 2013; Weller et al., 2008). Whilst some studies excluded people with current/past psychopathology (e.g, substance use, depression etc.), whether or not this included ED is not specified (Bongers et al., 2015; Daniel et al., 2013; Davis et al., 2011; Eisenstein et al., 2015; Jarmolowicz et al., 2014; Lawyer et al., 2015; Privitera et al., 2015). Other studies did not exclude people based on current/past psychopathology (Budría et al., 2010; Buono et al., 2015; Nederkoorn et al., 2006; Price et al., 2016).

In the first study of TD in obesity Nederkoorn et al. (2006) reported no difference in rates of TD between obese and healthy weight females and this finding has been replicated across males and females more recently (Bongers et al., 2015; Buono et al., 2015; Daniel et al., 2013; Eisenstein et al., 2015; Hendrickson and Rasmussen, 2013; Kulendran et al., 2016). However, after controlling for IQ, age and income, Weller et al. (2008) suggested that obese

women (but not men) have increased rates of TD. Similarly, seven other studies in obesity suggest that both obese males and females display increased rates of TD compared to controls (Bickel et al., 2014b; Garza et al., 2016; Jarmolowicz et al., 2014; Lawyer et al., 2015; Price et al., 2016; Schiff et al., 2015; Simmank et al., 2015).

Obese individuals classified as 'food addicts', compared to obese non-food addicts, also display increased TD (Davis et al., 2011). In contrast, one recent study, although confounded by its focus on depressive symptoms, reported that HC display increased TD compared to obese individuals (in relation to dessert foods only; Privitera et al., 2015). Interestingly, obese people who have been surgically treated for morbid obesity and are part of a weight control program (i.e. attend a nutrition/weight maintenance group) demonstrate reduced TD and thus a greater ability to delay gratification than HC (Budría et al., 2010). The authors attribute (but do not quantify) improved awareness of condition and commitment to recovery to the reduced rates of TD found in this particular group of obese individuals.

3.3 Neuroimaging findings (see Table 2)

3.3.1 Anorexia Nervosa (2 studies)

Two studies explored the neural correlates of TD in acute and remitted AN. Currently ill AN participants (within the first week of inpatient admission) showed reduced recruitment of regions implicated in reward processing, namely the striatum and dorsal anterior cingulate (dACC) during LL choices compared to SS choices. No choice-related differences were observed in the HC group. Following weight-restoration in AN (although still exhibiting significant ED psychopathology), greater recruitment of the striatum, dACC and DLPFC during LL choices were exhibited in comparison to both neural activity at admission and to that in HC. However, after controlling for subjective differences in choice difficulty, only the differences in striatal activation remained statistically significant (Decker et al., 2015). The increased recruitment of the striatum, dACC and DLPFC following weight restoration in AN

was associated with the normalisation of TD behaviour (i.e. increased preference for SS rewards; Decker et al., 2015).

Data from Wierenga et al. (2015) suggest that unlike in HC, hunger and satiety do not affect the neural correlates of TD in people remitted from AN. In HC, hunger increased activation of reward circuitry (e.g. dorsal anterior caudate, rostral and dACC, right striatum) while satiety increased cognitive control activity during SS choices (e.g. insula, ventrolateral PFC (VLPFC)). This pattern did not occur in individuals remitted from AN – i.e. hunger was not associated with increased activity of regions involved in reward valuation. When satiated, people who had remitted from AN activated reward valuation circuitry more than HC and regardless of metabolic/energy state, showed increased recruitment of the middle frontal gyrus (MFG) during SS choices. This suggests that compared to HC, individuals remitted from AN, exhibit atypical and heightened reward responses (when satiated) and more generally, elevated levels of cognitive control in relation to immediate choices.

3.3.2 Obesity (6 studies)

Four recent studies have examined associations between neural activity and TD behaviour in obesity. Two studies using the same sample, found that activity in frontoparietal regions (including the MFG, superior frontal gyrus (SFG) and inferior parietal lobe (IPL)) during difficult TD choices (i.e. choices closer to the participant's indifference point), was negatively correlated with TD (Stoeckel et al., 2013) and with weight gain up to 3 years later (Kishinevsky et al., 2012). In other words, reduced frontoparietal activity was associated with choice impulsivity and subsequent weight gain.

In line with these data, reduced TD (and thus better impulse control) prior to engaging in a 12-week low calorie diet predicted subsequent weight loss in obese individuals. This improved impulse control (i.e. reduced rates of TD) and dietary success were associated with stronger activity in the DLPFC and functional connectivity between control regions (DLPFC)

and regions signalling reward (e.g. the ventromedial PFC, (VMPFC; Weygandt et al., 2013). The same group replicated this finding (reduced TD associated with better weight maintenance) and found that control-related DLPFC activity (i.e. activity in the right SFG during difficult TD choices) was also associated with successful weight maintenance (Weygandt et al., 2015). Overall, these studies have shown that recruitment of circuitry involved in cognitive control was related to both rates of TD and dietary success in obese individuals.

In the only study to use positron emission tomography (PET) to explore TD in relation to ED and obesity, Eisenstein et al. (2015) reported a positive relationship between striatal D2 receptor binding/increased striatal dopamine transmission and rates of TD in obese (but not non-obese) individuals. This was seen in the absence of any behavioural TD differences between obese and non-obese individuals, nor were there any relationships between striatal D2 receptor binding and BMI or body fat. This suggests that striatal dopaminergic signalling may influence the subjective value of rewards specifically in obese individuals.

4. DISCUSSION

This review summarises and integrates existing behavioural and neuroimaging data on TD in ED and in obesity. In relation to our aims, we found that i) compared to controls, people with ED display both increased (BN, BED) and decreased/comparable (AN) rates of TD. However data in ED are very limited (only 4 studies in AN, 1 in BN and 5 in BED) and mixed (in the case of AN). Data in obesity are more comprehensive, but also mixed. However, a greater proportion of studies indicate increased rates of TD in people who are obese (compared to HC); ii) no study has compared TD between ED diagnoses and obesity; however when comparing rates of TD across studies, currently ill (but not weight-restored) AN has been associated with reduced TD (i.e. an increased ability to delay gratification)

whilst increased TD (i.e. choice impulsivity) is typically reported in BN, BED (albeit in minimal data) and often in obesity; iii) activity in/connectivity between reward valuation (e.g. striatum, dACC, VMPFC) and cognitive control circuitry (e.g. DLPFC) is implicated in TD and finally, iv) TD tasks used within this field vary markedly, primarily in relation to the presentation/framing of rewards and methods of analysis.

4.1 Theoretical implications

4.1.1 Anorexia Nervosa

Our findings have implications for the understanding of ED and obesity. Psychiatric conditions considered to date (e.g. addictive disorders, ADHD, schizophrenia, MDD etc.) are associated with increased rates of TD (Chantiluke et al., 2014; Demurie et al., 2012; Heerey et al., 2007; Pulcu et al., 2014; Reynolds, 2006). Whilst one study, limited by age differences between groups, found no difference in rates of TD between currently ill AN and HC (Ritschel et al., 2015), others suggest a unique neurobehavioural profile in the ill/underweight state of AN (i.e. reduced rates of TD; Decker et al., 2015; Steinglass et al., 2012). Only one other study has reported similar findings – individuals with OCPD display reduced rates of TD compared to both HC and individuals with obsessive compulsive disorder (Pinto et al., 2014). It is of note that OCPD is often comorbid with AN (Serpell et al., 2002) and that reduced TD in people with OCPD is associated with perfectionism and rigidity, i.e. traits characteristic of AN (Pinto et al., 2014). Therefore, in AN and OCPD, an excessive capacity to delay reward could be a shared underlying neuropsychological mechanism. However, whilst data reviewed here suggest that any reduced rates of TD in AN may normalise with recovery/weight restoration (Decker et al., 2015; Wierenga et al., 2015), these individuals still exhibit worse ED psychopathology than HC. Therefore, reduced rates of TD in the ill state of AN could equally be a marker of undernutrition, rather than indicative of specific AN psychopathology.

Although data are mixed and limited, considering stage-of-illness based differences in TD behaviour alongside related neuroimaging data, may be useful in understanding the pathogenesis of AN. In currently ill AN, reward related neural activity is reduced, yet activity in cognitive control regions is comparable to HC during LL choices (Decker et al., 2015). This suggests that people with AN do not overvalue nor are they driven by excessive cognitive control in relation to delayed choice options (argued to be further weight loss in AN; Steinglass et al., 2012). The unusual preference for LL rewards (i.e. reduced TD, reported in 2/3 studies) and related phenotypical 'self-controlled' symptoms of AN may therefore not be a result of the over-evaluation and pursuit of long-term goals (i.e. weight loss) but rather be habit driven (i.e. restricting food in order to reduce negative emotions), which might be particularly useful (e.g. energy conserving) in the underweight state of AN. This notion may also explain the differences in rates of TD reported between AN-R and AN-B/P (Decker et al., 2015; Steinglass et al., 2012), as the latter (i.e. AN-B/P) would rely on more 'impulsive' habitual symptoms (e.g. binge eating) and thus a relative preference for SS rewards. As mentioned, when exacerbated by the physical effects of starvation, these behaviours are argued to become inherently reinforced, habitual and compulsive (Park et al., 2014; Steinglass and Walsh, 2006; Walsh, 2013). Frederick et al. (2002) argue that compulsivity is a key component of TD, so data suggesting reduced TD in people acutely ill with AN and those with OCPD (Pinto et al., 2014) may reflect the rigid and compulsive nature of these illnesses, rather than reduced choice impulsivity per se.

Following weight restoration (but not necessarily improved psychopathology) in AN, the normalisation of discounting behaviour (preference for SS rewards) is associated with increased activation in both reward (e.g. striatum) and cognitive control (e.g. DLPFC) regions during LL choices (Decker et al., 2015). This may reflect improved cognitive resources following weight gain to properly evaluate choices. Increased valuation and 'top-down'

control over the pursuit of actually higher valued (rather than habit driven) delayed rewards (i.e. long-term benefits of recovery) may enable compulsive behaviours (e.g. food restriction and thus a habitual resistance to SS choices) to be overridden. However, these implications are speculative and based on minimal, mixed data. More work is needed to explore the unique concept of reduced, state-related TD in AN and what this may represent (i.e. reduced impulsivity or rather compulsive features of the illness). Future studies should consider confounding factors such as age, illness duration and a careful definition of remission/recovery, when examining TD in AN.

4.1.2 Bulimia Nervosa and Binge Eating Disorder

In regards to BN and BED, limited behavioural data exist yet they support the notion of increased rates of TD in these two ED. However, only one study in BN exists. Moreover, all BED studies were in overweight/obese individuals, two of which reported comparable rates of TD in obese people both with and without BED (Davis et al., 2010; Mole et al., 2015). As reviewed here, obesity is often associated with increased rates of TD. Given all studies of BED are in obese people and that obesity/BED are often indistinguishable in terms of TD, overall findings in BED may be biased. Additionally, normal weight people with BED are proposed to be behaviourally distinct from obese people with BED (Goldschmidt et al., 2011). No conclusions can be drawn about this subset of individuals with BED.

However, increased rates of TD in BN and obese individuals with BED are consistent with findings across many psychiatric conditions (Chantiluke et al., 2014; Demurie et al., 2012; Heerey et al., 2007; Pulcu et al., 2014; Reynolds, 2006). Whilst such findings are often generalised to reflect heightened levels of impulsivity within these conditions, Story et al. (2015) uses a biopsychosocial approach to explore the goals and motivations for discounting future rewards. Some of the key motivations for discounting future rewards are proposed be the opportunity costs of delay, uncertainty associated with future outcomes and the cognitive

costs of resolving uncertainty. In the context of an individual's life history/previous experiences and the choice environment, these motivations and their underlying neurobiological/physiological mechanisms, may offer an alternative explanation to heightened discounting of future rewards in people with a psychiatric condition (Lempert et al., 2015; Story et al., 2015). Alternatively, given our interpretation of the findings reviewed in AN here and the multidimensional nature of TD argued by Frederick et al. (2002), increased rates of TD in BN and BED could equally represent the compulsivity and/or poor inhibitory control processes proposed to underlie binge eating and/or purging symptoms in these disorders (Bartholdy et al., 2016; Guillaume et al., 2015).

Relatedly, individuals both with and without BED (who are obese) also showed comparable rates of TD to abstinent alcohol-dependent individuals (Mole et al., 2015). Other findings of the association between increased TD and binge eating in heavy drinkers further supports the notion of trans-disease neurobehavioral processes across ED symptoms and addictive disorders (Davis and Claridge, 1998). Interestingly, rates of TD in people with and without BED are distinguishable in overweight individuals (Manasse et al., 2015a; Manasse et al., 2015b) yet indistinguishable in people who are obese (Davis et al., 2010; Mole et al., 2015). This suggests that TD could act as a gauge of worsening decision-making abilities with the development of obesity.

4.1.3 Obesity

Data regarding TD in obese people are mixed. The largest proportion of studies (e.g. 44%) report increased rates of TD in obesity compared to controls, however two studies suggest the opposite (e.g. reduced rates of TD) and many studies (39%) report no differences in TD between HC and obese people.

A number of inconsistences across studies of obesity, including sampling criteria and heterogeneity in TD tasks/analyses, are likely to contribute to such disparity in findings.

Firstly, many studies included overweight individuals in their 'obese' group (Buono et al., 2015; Daniel et al., 2013; Jarmolowicz et al., 2014; Price et al., 2016). Such studies may be examining TD in a different and perhaps less severe sample of overweight individuals than the majority of studies which only include obese people (i.e. BMI > 30 kg/m^2). Relatedly, most obesity studies reviewed here did not specify if they excluded/controlled for comorbid psychiatric conditions, including BED, in their analysis of TD. Given the high rates of co-morbidity between obesity and BED and the worse psychopathology in this group (Villarejo et al., 2012) findings of increased rates of TD in obesity studies that do not account for previous/current ED/psychiatric conditions may not be attributable to obesity alone.

Importantly, many studies report differences in TD after controlling for factors such as sex, education level and socio-economic status (Budría et al., 2010; Jarmolowicz et al., 2014; Lawyer et al., 2015; Price et al., 2016; Weller et al., 2008). However, studies that did not control for these factors report no differences in rates of TD (Bongers et al., 2015; Daniel et al., 2013; Eisenstein et al., 2015; Hendrickson and Rasmussen, 2013; Nederkoorn et al., 2006; Privitera et al., 2015). Finally, different reward reinforcers are discounted more/less steeply (Odum et al., 2006). The variety of reinforcers used (e.g. money, foods, massage time etc.) in studies of TD in obesity, are likely to have contributed to inconsistencies in findings.

Interestingly, food addiction in people who are obese is associated with BED comorbidity and increased rates of TD respectively (Davis et al., 2011; Manasse et al., 2014), further supporting the notion of TD being associated with addictive psychopathology in relation to ED/obesity. However, whilst Eisenstein et al. (2015) replicate previous findings regarding dopamine and TD in healthy individuals (Pine et al., 2010), their data oppose the finding of reduced striatal dopamine D2 receptor binding and increased rates of TD in addictive disorders (Ballard et al., 2015; Oberlin et al., 2015). Therefore, whilst behaviourally

obesity and addiction may both be associated with increased rates of TD, this may not arise from the same neurobiological mechanisms.

Although TD behaviour is typically argued as a predetermined personality trait (Odum, 2011), obese individuals post-bariatric surgery who are trying to control their weight show reduced rates of TD compared to controls (Budría et al., 2010). Not only is this the opposite of the increased rates of TD often reported in people who are obese, it also mirrors findings reported in currently ill AN patients (Decker et al., 2015; Steinglass et al., 2012). When considered alongside neuroimaging data, these findings suggest that symptoms related to increased/decreased rates of TD in obesity/acute AN respectively, may both be remedied by improved cognitive control mechanisms gained through treatment programs/weight restoration (Decker et al., 2015; Weygandt et al., 2013).

Neuroimaging studies in both AN (discussed previously) and obesity support this. In people who are obese, reduced activation in brain areas associated with cognitive control is associated with increased rates of TD (Stoeckel et al., 2013; Weygandt et al., 2015) and predicts future weight gain (Kishinevsky et al., 2012; Weygandt et al., 2013). This is perhaps unsurprising, given the evidence suggesting that the ability to exert self-control over immediate temptations relies on activity in the right PFC (Knoch and Fehr, 2007) and that impulse control in relation to food-specific tasks relies on the DLPFC (Hare et al., 2009; Hare et al., 2011). However, the localisation of key regions associated with what is quite a complex behaviour is inherently difficult, suggesting that the neural correlates of intertemporal choice behaviour are not isolated to one specific region. This argument is supported by the findings of Weygandt et al. (2013), suggesting that reduced connectivity between the DLPFC and VMPFC is associated with increased rates of TD. Given that increased TD was found to be associated with poorer dietary success in this study, impaired functional connectivity between frontal brain areas, crucial in cognitive control mechanisms,

could be a key contributor to the development and maintenance of obesity and, moreover, a predictor of treatment response.

4.1.4 Temporal discounting: trait or state?

As mentioned, an important finding from the studies reviewed here is that, despite longstanding arguments that TD is a fixed personality trait (Odum, 2011), TD in ED and in obesity seems to vary according to a number of factors. Rates of TD in people who have an ED/are obese may fluctuate according to illness stage (e.g. currently ill and weight-restored AN; Decker et al., 2015; Steinglass et al., 2012; Wierenga et al., 2015), metabolic state (e.g. hedonic hunger, hunger/satiation; Manasse et al., 2015a; Wierenga et al., 2015) and concern over controlling weight in obesity (Budría et al., 2010).

Other findings support the idea that TD is changeable. Relevant to ED and obesity, healthy individuals who drank a soft-drink containing artificial sweetener or glucose showed an increase and decrease in rates of TD, respectively (Wang and Dvorak, 2010). Moreover, in both HC and people with addictive disorders, physiological states (e.g. sleep and cigarette deprivation, alcohol consumption, methylphenidate administration), framing effects (e.g. episodic future thought), conditioned stimuli (e.g. viewing attractive/unattractive people) and therapeutic interventions (e.g. working memory training, neuromodulation) have been shown to modulate rates of TD (for a review see Koffarnus et al., 2013). However, these studies report only short-term changes to TD (e.g. immediately following manipulation/training). There is currently no evidence to support the long-term stability of changes to TD following such interventions, which impedes our understanding of the state/trait nature of TD.

4.2 Clinical implications

4.2.1 Psychoeducation and outcome prediction

Findings from this review have implications for psychoeducation, prognosis and treatment in ED and in obesity. Evaluating the concept of TD in individuals with ED and/or

obesity and providing them with normative feedback may promote understanding of neuropsychological mechanisms underpinning or contributing to symptomatology and provide a helpful rationale for the focus of subsequent treatment. Moreover, TD has been shown to predict treatment outcomes in addiction (Bickel et al., 2014a); data reviewed here support its predictive utility in obesity (Weygandt et al., 2013; Weygandt et al., 2015) but this is yet to be explored in relation to ED.

4.2.2 Treatment

Treatments that target neurobehavioural processes related to TD (e.g. dysregulated frontostriatal circuitry, attentional biases, impaired executive function, etc.) may improve clinical outcomes (Appelhans et al., 2016; Voon, 2015). For example, two non-invasive neuromodulation techniques, repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS), applied to the DLPFC, have been shown to alter rates of TD (direction depends on stimulation parameters; Cho et al., 2010; Cho et al., 2012; Figner et al., 2010; Hecht et al., 2013) and improve clinical outcomes in ED and obesity (McClelland et al., 2013; Val-Laillet et al., 2015). In an attempt to modulate the neurocircuitry proposed to underpin self-regulation difficulties in ED (Friederich et al., 2013; Kaye et al., 2011; Kaye et al., 2009; Marsh et al., 2009a), we have shown that both rTMS and tDCS lead to a reduction in rates of TD (i.e. individuals discount future rewards less) as well as symptom improvements in both AN and BN, respectively (Kekic et al., submitted; McClelland et al., 2016). However, these reported changes to TD are immediately following a single session of rTMS/tDCS. Longitudinal research of rTMS/tDCS applied therapeutically (i.e. typically 20-40 sessions) is needed in order to ascertain whether such interventions are capable of inducing sustained changes to discounting behaviour.

Alternatively, attentional/cognitive bias modification training (ABMT/CBMT) aims to target automatic attentional processes. Altering temporal attention during TD tasks has

been shown to reduce the discounting of future rewards (Radu et al., 2011) and ABMT/CBMT using salient cues (e.g. food/body images) have been found to offer potential in the treatment of ED and obesity (Boutelle et al., 2014; Boutelle et al., 2016; Brockmeyer et al., 2015; Kemps et al., 2014; Renwick et al., 2013). Other treatments, targeting TD-related processes include Cognitive Remediation Therapy (CRT) which aims to improve cognitive flexibility and working memory (Basile and Toplak, 2015; Wesley and Bickel, 2014), mindful decision making and inhibition training (Forman et al., 2016). Whilst the effects of these interventions on intertemporal choice behaviour are yet to be reported, these interventions have shown potential in treating ED and obesity (Danner et al., 2015; Raman et al., 2014).

4.3 Methodological considerations

The implications emerging from this review must be interpreted with caution. As indicated here and elsewhere, TD tasks are widely heterogeneous (MacKillop et al., 2011). For example, discounting rates for real and hypothetical rewards are argued to be analogous (Locey et al., 2011) and ecologically valid; however, results from studies using both may not be directly comparable. Tasks also vary from brief surveys/questionnaires to more sophisticated and personalised computerised assessments. The latter are a more thorough and precise measurement of TD, yet a greater proportion of studies using questionnaire-based assessments reviewed here report differences in TD behaviour; the robustness of these findings are questionable. In addition, the framing of rewards also differs across studies and is likely to affect discounting outcomes; tasks that employ both accelerate and delay sets may be more informative and thus favourable (DeHart and Odum, 2015).

Whilst using monetary rewards in TD tasks for people with an ED/obesity overcomes difficulties in the interpretation of responses to symptom-provoking rewards, e.g. food (Steinglass et al., 2012), monetary rewards are discounted differently (i.e. less steeply) than

directly consumable rewards (Estle et al., 2007; Odum and Rainaud, 2003). Using ED/obesity salient stimuli (i.e. high/low calorie foods) has proved useful in differentiating individuals with BED (Manwaring et al., 2011) and therefore could produce equally informative, illness-specific findings in other ED and in obesity.

The way in which TD is quantified and analysed varies. The use of simple hyperbola (*k*) is thought to be associated with interpretative and statistical difficulties, whilst AUC is argued to be a theoretically neutral, less sensitive and thus more favourable option for investigations with quantitative, inferential statistics (Myerson et al., 2001). However, in the papers reviewed here, a greater proportion of studies reported differences in rates of TD using *k* as an outcome measure of TD, which, given its statistical robustness, may therefore be a more favourable outcome measure.

Related to methods of analyses, whilst some studies ran preliminary analyses to assess group differences in critical confounding factors, e.g. age, sex, IQ, education or income, (Green et al., 1996), fewer than 30% of studies controlled for these in their analysis of TD. In those that did, group differences in TD behaviour persisted, demonstrating the robustness of these findings. It is recommended that these factors are accounted for in the analyses of future TD studies.. Moreover, whilst some studies excluded participants with psychiatric comorbidities, only 15% of studies controlled for co-morbid psychopathology (e.g. depressive symptoms) in their TD analyses. ED and obesity are often associated with co-morbid psychopathology and this has been proposed to be potential confounder in evaluating neuropsychological performance (Abbate-Daga et al., 2015). Since most psychiatric conditions are associated with increased rates of TD, this limits the specificity of many findings reviewed here to ED and obesity psychopathology alone.

4.4 Conclusions

This review provides understanding of an important neurobehavioral process, namely intertemporal choice behaviour, in people with ED and obesity. In relation to our primary aims, limited and mixed evidence reviewed here suggest that compared to HC, people with BN, BED and obese individuals, display increased choice impulsivity. Data in AN are also limited and mixed. Current (but not remitted) AN has been associated with both normal and an exaggerated ability to delay gratification, the latter could reflect the compulsive aspects of the illness and/or be attributable to the undernourished state of acute AN. Neuroimaging data (in AN and obesity) implicate brain regions involved in reward valuation, cognitive control and their functional connectivity, in regards to TD behaviour and clinical outcomes. Unfortunately, the wide heterogeneity across TD tasks used within this field limits comparability and generalisability of findings.

The behavioural differences in TD reviewed here could support a spectrum model of self-regulation difficulties in ED and obesity. However, considered alongside neuroimaging data and stage-of-illness based differences in TD, findings implicate a multidimensional nature to TD. Specifically, TD tasks may capture dimensions of intertemporal choice behaviour that are highly relevant to both ED and obesity (e.g. impulsivity, compulsivity and inhibitory control) and thought to be underpinned by impaired frontostriatal circuitry. Moreover, the fact that both TD behaviour and its neural correlates in ED and obesity seem to vary according to illness stage and metabolic state, raises questions regarding the state/trait nature of TD in these conditions. The idea that TD processes may fluctuate according to such factors, has important implications for assessment and treatment of ED and obesity.

4.5 Future directions

This review provides an evaluation of the most informative ways to assess TD behaviour and its neural basis in ED and obesity. Standardising the measurement of TD (e.g.

using hyperbolic functions) and ensuring relevant factors are accounted for (e.g. age, IQ, income, education and comorbid psychopathology) will be of most use in ensuring comparable future research. Determining whether TD measures a unitary construct, or in fact captures a number of sub-dimensions relevant to ED and obesity warrants further examination because it remains unclear. This could be achieved via transdiagnostic research involving neurobehavioural tasks relevant to TD, to conceptually deconstruct intertemporal choice behaviour, along with neuroimaging, to elucidate neural mechanisms. Longitudinal research is also needed to understand the state/trait related nature of TD in ED and obesity. This knowledge could then be incorporated into personalised treatment plans that target specific or broader self-regulatory control difficulties in ED and obesity.

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7. FIGURE CAPTIONS

Figure 1. Schematic representation of temporal discounting

Figure 2. PRISMA flow diagram

8. TABLES

Table 1. Behavioural temporal discounting findings in eating disorders and obesity

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Studies in Anorexia Nervosa							
Decker et al. (2015)	98	T1: pre-treatment AN inpatients (n=59, 2 male) & HC (n=39, 2 male). T2: weight-restored AN (n=43) & HC (n=31). AN groups included people with anxiety & depressive disorders.	Monetary; real (paid according to preference on one randomly selected trial).	Computerised Two sets; accelerate set: SS variable, LL fixed; delay set: SS fixed, LL variable. SS option available now or in 2 weeks; LL option available 2 or 4 weeks. 72 binary choices.	k	T1: AN < HC; AN-R < HC; AN-BP = AN-R; AN-BP = HC. T2: AN = HC; AN-R = HC; AN-BP = AN-R; AN-BP = HC. From T1 to T2 significant change in TD in AN, AN-R and AN-BP groups. At T1 AN showed slower response times for SS choices than LL; at T2 AN showed slower responses for LL choices than SS. T1 HC = T2 HC.	Change in TD seen in AN not due to their proximity to discharge. TD not associated with BMI, illness duration or symptoms. The primary findings remained the same when age and IQ were included in the model HC were matched for age, sex and ethnicity. Comorbid psychopathology not controlled for in analyses.
Ritschel et al. (2015)	124	Females with AN 'acAN' (n=34) who were reassessed after partial weight restoration (n=21), females recovered from AN 'recAN' (n=33) & HC (n=53). 9.1% of acAN group had comorbidities: depression (6.1%), anxiety (2.9%) & OCD (2.9%). 27.3% of recAN had comorbidities: depression (24.2%) & OCD (3%).	Monetary; hypothetical.	Computerised SS immediate and fixed; LL variable. 5 delays (10-180 days). 50 binary choices.	k & AUC	acAN = recAN = HC; AN-R = AN-BP. acAN-T1 = acAN-T2 following weight restoration.	Adolescents in sample: acAN M=15.29yrs, recAN M=21.67yrs, HC M=18.75yrs. acAN significantly younger than recAN and HC. No relationship between TD and BMI, age of onset or duration of AN. In recAN, TD positively associated with body dissatisfaction and ED pathology. Comorbid psychopathology not controlled for in analyses

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Steinglass et al. (2012)	64	Female underweight AN (n=36) & HC (n=28). AN group included 6 individuals with prior history of substance abuse.	Monetary; real (1 in 3 chance of receiving gift card to value of random choice preference).	Computerised Two sets: accelerate set: SS immediate & variable, LL fixed; delay set: SS immediate & fixed, LL variable. Fixed 3 month delay period. 26 binary choices.	Separate discount factor for each set; δ	AN < HC (particularly AN-R subtype); AN-BP = HC; AN-R = AN-BP.	In AN, BMI positively associated TD. Delay set and history of substance abuse associated with increased TD. When those with substance abuse history removed from analyses, effects persisted. No other comorbid psychopathology controlled for in analyses.
Wierenga et al. (2015)	40	Females remitted from AN 'remAN' (n=23) & HC (n=17). remAN group: no current psychiatric comorbidities or substance abuse within 3 months prior. Historical comorbidities included: depressive disorder (n=17), anxiety (n=8), OCD (n=4) & substance dependence (n=3).	Monetary; real (paid according to preference on one randomly selected trial).	Computerised SS/LL reward paradigm unclear. 32 binary choices.	k	remAN = HC.	HC responded more slowly when satiated than when hungry. No difference in response time between remAN & HC when satiated. No difference in response time in remAN between hungry vs satiated state. Comorbid psychopathology not controlled for in analyses.
Studies in Bulimia Nervosa							
Kekic et al. (2016)	96	BN (n=39) & HC (n=53). BN group: people not excluded based on comorbidities.	Monetary; hypothetical.	Computerised Two sets: accelerate set: SS immediate & variable, LL fixed; delay set: SS immediate & fixed, LL variable. Fixed 3 month delay period. 80 binary choices.	Separate discount factor each set; δ	BN > HC.	Effects persisted when depression, anxiety & stress scores controlled for in analysis.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Studies in Binge Eating Disorder/loss-of-control eating							
Davis et al. (2010)	209	Obese women (n=73), obese women with BED (n=65), and HC (n=71). BED group included people with depression (9.2%)	Monetary; hypothetical.	Computerised SS varied; LL fixed ($100). 5 delays (2-730 days). Number of binary choices NR.	Indifference points	Generally, obese > HC; obese with BED > HC; obese = obese with BED.	When education differences accounted for (HC more educated than BED & obese) findings no longer significant. Comorbid psychopathology not controlled for in analyses.
Manasse et al. (2014)	80	Overweight/obese females with loss of control (LOC) eating (n=18) & without LOC eating (n=62). Co-morbidities not considered or assessed (apart from purging behaviours).	Monetary; hypothetical.	Computerised SS varied & immediate; LL fixed ($1000). 8 delays (6 hours-25 years). 240 binary choices.	AUC	LOC = no LOC.	LOC group had higher rates of depression. Age, IQ and depression were included as covariates. Apart from depression, no other comorbid psychopathology was controlled for in analyses.
Manasse et al. (2015a & 2015b)	74	Overweight/obese women with full/subthreshold BED (n=31) & overweight/obese control (n=43). BED group: no history of BN; control group: no history of ED.	Monetary; hypothetical.	Computerised SS varied & immediate; LL fixed ($1000). 8 delays (6 hours-25 years). 240 binary choices.	AUC	Overweight/obese BED > overweight/obese control.	No relationship between TD and binge frequency. Full-BED & subthreshold-BED participants did not differ on TD. TD associated with likelihood of BED in presence of low hedonic hunger. BED group had higher rates of depression. Age, IQ and depressive symptoms were included as covariates. Differences no longer significant when depression scores controlled for. No other comorbid psychopathology was controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Manwaring et al. (2011)	90	Obese women with BED (n=30), obese women without BED (n=30) & HC (n=30). No participants had psychosis or severe depression within past 3 months.	Monetary, food, sedentary activity & massage time; hypothetical.	Computerised SS immediate & varied; LL fixed. 5 delays (1 week-3 years). 30 binary choices.	AUC	Food: Obese with BED > HC; Obese with BED > obese without BED & HC; obese without BED> HC. Money: obese with BED > HC; obese with BED > obese without BED (trend); obese without BED= HC. Sedentary activity: obese with BED > HC; obese with BED > obese without BED; obese without BED = HC. Massage time: obese with BED > HC; obese with BED > obese without BED; obese without BED > HC.	All groups discounted food rewards more steeply than other non-monetary reward types. Probability discounting task also completed. Comorbid psychopathology not controlled for in analyses.
Mole et al. (2015)	180	Obese with BED (n=30, 13 male) & matched HC (n=30, 13 male); obese without BED (n=30, 19 male) & matched HC (n=30, 19 male), abstinent alcohol-dependent (n=30, 18 male) & matched HC (n=30, 18 male). No participants had current depressive episode, substance use disorder & no history of psychiatric disorder.	Monetary; real (1 in 6 chance of winning their preferences on randomly selected trial).	Questionnaire (MCQ) SS immediate & variable; LL variable. Delays ranged 1 week-6 months. 27 binary choices.	k	Obese with BED & obese without BED > HC. Abstinent alcohol-dependent = obese with BED = obese without BED.	In obese people with & without BED no correlation between TD and BMI, Binge Eating Scale or depression scores. BED and alcohol-dependent groups had higher rates of depression than HC. Increased depression scores, or any other psychopathology were not controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Studies in Obesity							
Bickel et al. (2014)	1163	Obese (n=263; 100 male) & non-obese (n=900; 415 male). Co-morbidities not considered or assessed (including ED).	Monetary; hypothetical.	Questionnaire SS & LL both varied. Delays varied (10-75 days). 21 binary choices.	k	Obese > non-obese.	Results reported for Me-Me condition only. Age, education, gender, income considered in regression. Probability discounting task also completed. Comorbid psychopathology not controlled for in analyses.
Bongers et al. (2015)	319	Obese (n=185; 54 male) & matched HC (n=134; 35 male). People with major psychiatric disorders excluded.	Monetary; hypothetical.	Computerised SS immediate & varied (€12-€988); LL fixed (€1000). 7 delays (2 weeks-10 years). 42 binary choices.	AUC	Obese = HC.	Obese group displayed increased rates of food craving, impulsiveness, and dietary/eating psychopathology. Binge-eating also more common in obese group. Comorbid psychopathology not controlled for in analyses.
Budria et al. (2012)	41	Obese people post obesity surgery and part of weight control group (n=26) & HC (n=15). Co-morbidities not considered or assessed (including ED).	Monetary; real (person randomly selected).	Questionnaire SS fixed (€300); LL varied (€303-€469). Fixed delay interval (6 months). 20 binary choices.	Discount rate (r)	Obese post-surgery < HC.	Controlled for personality traits & socio-economic status. Gender not reported. Comorbid psychopathology not controlled for in analyses.
Buono et al. (2015)	38	Overweight/old (n=10; 2 male, 45-55yrs), overweight/young (n=10; 2 male, 18-27yrs), normal weight/old (n=8; 5 male, 45-55yrs), normal weight/ young (n=10; 0 male, 18-27yrs).	Monetary, hypothetical.	Computerised Descending from $1000 to $10. 7 delays (1 week - 10 years). 175 binary choices.	AUC	Overweight = HC.	Overweight groups include people who are obese. Significant interaction between age and weight: older overweight individuals < young overweight individuals. Main effect of age: old < young. No difference between young and old normal weight.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
		Co-morbidities not considered or assessed (including ED).					Comorbid psychopathology not accounted for in analyses.
Daniel et al. (2013)	48	Overweight/obese women (n=24) & lean women (n=24). No current depression or psychopathology as exclusion criteria.	Monetary, hypothetical.	Index cards SS immediate & varied (decreased from $100 to $1); LL fixed ($10 and $100). 7 delays (1 day - 2 years). 26 binary choices.	AUC	Overweight/obese = HC.	Also investigated effects of episodic future thinking on TD and task differed due to that component. Controlled for vividness of imagery, and scores on the ZTPI and BIS/BAS. Comorbid psychopathology not accounted for in analyses.
Davis et al. (2011)	72	Obese food addicts (n=54, 18 male); obese non-food addicts (n=18, 5 male). Exclusion: current diagnosis of any psychotic or substance abuse disorder.	Monetary; hypothetical.	Computerised SS immediate & varied; LL fixed ($100). 5 delays (2-730 days). Number of binary choices NR.	Total of indifference points for all 5 delays.	Obese food addicts > obese non-food addicts.	More of the food addicts had BED, had higher depression scores & greater proportion childhood ADHD, more impulsive, more binge eating, hedonic eating, emotionally driven eating, cravings and snacking. Comorbid psychopathology not controlled for in analyses.
Eisenstein et al. (2015)	47	Obese adults (n=27, 4 male) & non-obese (n=20, 5 male). Exclusion: any history of psychiatric diagnosis, including substance abuse/dependence.	Monetary; hypothetical.	Computerised SS immediate & varied; LL fixed ($500). 5 delays (1 week-2 years). 25 binary choices.	AUC	Obese = HC.	Within total sample and separate groups BMI not correlated with TD. Analyses accounted for age, education level, sex, and ethnicity. Comorbid psychopathology not controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Garza et al. (2016)	478	Underweight (n=8), healthy weight (n=195), overweight (n=137) and obese (n=132); 149 male. Comorbidities not considered or assessed.	Monetary; hypothetical.	Computerised SS immediate & varied; LL fixed ($1000). 7 delays (1 day-25 years). 42 binary choices.	AUC	Obese > underweight, healthy weight & overweight.	Sex, age, marital status, income, education level measured and not associated with TD. Not included as covariates in TD analysis. Comorbid psychopathology not controlled for in analyses.
Hendrickson and Rasmussen (2013)	287	Obese (n=NR) and HC (n=NR). People with possible ED within past 2 years excluded from all analyses.	Food & monetary; hypothetical.	Computerised Food: SS immediate & varied; LL fixed (10 bites). 5 delays (1-20 hours). Number of binary choices NR. Money: SS immediate & varied; LL fixed ($10). 5 delays (1-365 days). Number of binary choices NR.	k & AUC	Obese = HC.	Individuals with higher percent body fat discounted food (but not money) more steeply than individuals with lower percent body fat. Only results for experiment 1 reported. Also report probability discounting and mindful eating intervention (experiment 2). Comorbid psychopathology not controlled for in analyses.
Jarmolowicz et al. (2014)	100	Obese/overweight (n=49, 30 male) & underweight/HC (n=51, 21 male). Excluded if current/past substance abuse, diagnosis of psychiatric illness.	Monetary, hypothetical.	Questionnaire (MCQ) SS immediate & variable; LL variable. Delays ranged 1 week – 6 months. 27 binary choices.	k	Overweight/obese > underweight/HC.	Findings remained significant after covarying for age, education and income. Body mass significantly correlated and predicted TD. Comorbid psychopathology not accounted for in analyses.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Kulendran et al. (2016)	105	Obese bariatric (n=45, gender NR), obese lifestyle management (n=20, gender NR) & HC (n=40, gender NR). Excluded if: has an ED or ADHD.	Monetary; real (one trial selected).	Computerised SS immediate: variable/fixed nature of SS/LL rewards NR. Number of delays and binary choices NR.	k	Bariatric obese = lifestyle obese = healthy.	Age, gender, education level not accounted for. No differences in TD based on sex. Comorbid psychopathology not controlled for in analyses.
Lawyer et al. (2015)	291	Obese (n=56, 19 male) & non-obese (n=235, 109 male). Comorbidities not considered or assessed.	Monetary; hypothetical.	Computerised SS immediate & varied ($20 increments); LL varied. 7 delays (1 day-25 years). Number of binary choices NR.	k	Obese > non-obese.	Controlled for age, sex and substance use. BMI significantly correlated TD. Obese significantly older. Also report probability discounting. Apart from substance use, no other comorbid psychopathology controlled for in analyses.
Nederkoorn et al. (2006)	59	Obese women (n=31) & HC women (n=28). Comorbidities not considered or assessed.	Monetary; hypothetical.	Format NR SS immediate and variable (€990-100); LL fixed (€1000). 7 delays (1 week-25 years). Number of binary choices NR.	k	Obese = HC.	Comorbid psychopathology not controlled for in analyses.
Price et al. (2016)	79	Overweight/obese (n=38, 16 male) & lean (n=41, 9 male) Comorbidities not considered or assessed.	Monetary; hypothetical	Computerised SS immediate & varied; LL fixed (£100) 9 delays (1 day – 1 year) Number of binary choices NR.	3 models; Q, sat*k* & *k*	Overweight/obese > lean	Results remained significant when age and gender accounted for in analysis. Comorbid psychopathology not controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
Privitera et al. (2015)	92	Undergraduate students (32 male): overweight (n=NR), obese (n=NR) & lean (n=NR). Participants with & without depression took part. No other comorbidities considered (including ED).	Food (dessert, fried food, fruit, vegetable); hypothetical.	Computerised SS immediate and variable; LL fixed (10 servings). Fixed delay (4 hours). Number of binary choices NR.	Mean DD score	With no depression, lean > obese (dessert foods only).	No differences in TD between BMI groups in regards to fruit and vegetables only. With severe depression, overweight/obese > lean (dessert and fried food only). Analyses controlled for self-reported ratings of hunger. Comorbid psychopathology not controlled for in analyses.
Schiff et al., (2015)	46	Obese (n=23, 5 male) & HC (n=23, 5 male). Excluded if: current/past ED or other psychiatric disorder, history of substance abuse.	Food, money & discount voucher; hypothetical.	Computerised SS immediate & varied; LL fixed (40 units). 6 delays (2 days-1 year). 30 binary choices.	k	Obese > controls (food only). Obese = controls (money & discount voucher).	Age, education and other factors not controlled for in analysis. Comorbid psychopathology not controlled for in analyses.
Simmank et al. (2015)	52	Obese (n=NR) and lean (n=NR); 26 male. Excluded if had current/past addictive disorder, history of mood disorders (e.g. anxiety and OCD), ED or high depressive/food addiction psychopathology.	Monetary; real (chance to win one of choices)	Computerised SS immediate and fixed; LL variable. 6 delays (1-12 months). Number of binary choices differed between subjects.	Quasi-hyperbolic model	Obese > lean.	Measured intelligence, education level, income, impulsivity and sensitivity to reward, however did not control for these in TD analysis. Comorbid psychopathology not controlled for in analyses.
Weller et al. (2008)	95	Obese (n=48, 19 male) & matched HC (n=47, 21 male). Excluded if: current/past smoker, substance	Monetary; hypothetical.	Computerised SS immediate & variable, LL fixed at £50k or £1k. 7 delays (2 weeks-10 years).	AUC	Obese women > HC women. Obese men = HC men. HC women = Obese men = HC men.	Remained significant when controlling for IQ, age and income. Comorbid psychopathology not controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Outcome	Primary findings: rate of temporal discounting	Other findings/comments
		abuse/gambling, or ED. No other comorbidities considered (e.g. depression).		42 binary choices.			

AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder; SS = smaller/sooner; LL larger/later; HC = healthy controls; NR = not reported; MCQ = monetary choice questionnaire; TD = temporal discounting; T1: time points 1; T2 = time point 2; yrs = years, ZTPI = Zimbardo Time Perspective Inventory; BIS/BAS = The Behavioral Inhibition and Behavioral Approach Scale, M = mean.

Table 2. Neuroimaging temporal discounting findings in eating disorders and obesity

Authors	N	Sample	Reward	Task characteristics	Scanning/analysis procedure	Primary findings: neural correlates of temporal discounting	Other findings/ comments
Studies in Anorexia Nervosa							
Decker et al. (2015)	46	Female inpatients with AN (n=25) and HC females (n=21). T1: pre-treatment AN inpatients (n=23, 2 excluded) & HC (n=21). T2: weight-restored AN (n=16) & HC (n=18). AN group excluded based on: history of bipolar, psychotic disorder, substance abuse in last 6 months. Anxiety and depressive disorders included. HC excluded based on: no current or past psychiatric illness.	Monetary; real (paid according to preference on one randomly selected trial).	Computerised Two sets; accelerate set: SS variable, LL fixed; delay set: SS fixed, LL variable. SS option available now or in 2 weeks; LL option available 2 or 4 weeks. 72 binary choices. Outcome: k.	2 scans: once within week of admission, one after weight restoration. Time between sessions matched for HC group. Analysis: Linear mixed-effects model exploring the interactions between fixed effects variables (choice, set, group, scanning session, repeated with and without including subjective value as a regressor to account for choice difficulty (i.e., harder trials = closer to indifference point)). T-tests compared difference in choice activity (LL minus SS) between groups.	Significant choice (SS/LL) x group x session interaction (bilateral striatum, dACC, right DLPFC, right parietal lobule). T1 (admission): HC: LL = SS; AN: LL < SS (dACC & striatum); AN < HC in LL choices (dACC & striatum). T2 (weight restoration): HC: LL < SS (dACC, right DLPFC, right parietal lobule); AN: LL > SS (striatum, dACC, right DLPFC). Longitudinal changes (LL-SS activity in striatum, dACC, right DLPFC, right parietal lobule): HC: T1 > T2; AN: T1 < T2. Normalisation of TD (i.e. increasing TD in AN) associated with increased activation in reward (striatum, dACC) and decision making (DLPFC, parietal cortex) regions during LL compared to SS trials.	After adding subjective value (choice difficulty) into the model, only differences in the striatum remained significant (no longer group/session differences in right DLPFC, dACC or parietal regions). Age and IQ included as covariates in the model. Comorbid psychopathology not controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Scanning/analysis procedure	Primary findings: neural correlates of temporal discounting	Other findings/ comments
Wierenga et al. (2015)	40	Females remitted from AN 'remAN' (n=23) & HC (n=17). No remAN had psychiatric comorbidities or history of alcohol/drug abuse/dependence 3 months prior to study. 17 had history of depressive disorder, 8 had history of anxiety, 4 had history of OCD and 3 had history of substance dependence.	Monetary; real (paid according to preference on one randomly selected trial).	Computerised SS variable/ variable. 3 SS delays (today, 2 weeks, 4 weeks); 2 LL delay (2 weeks, 4 weeks after SS). Difference between SS & LL rewards varied across 6 percentages (3-35%). 30 binary choices. Outcome: k.	2 scans, 24 hours apart: one in hungry state (fasted 16 hours) & one in fed state (consumed standardised breakfast). Analysis: group x visit linear mixed effects analysis. Two general linear models were conducted to assess reward valuation response and cognitive control response separately: Reward valuation: model included only decisions including immediate SS choices. ROIs in 'Valuation circuitry': VS, dorsal ACC, rostral ACC (VMPFC), PCC, dorsal caudate. Cognitive control: model included all decision trials. ROIs in 'Cognitive circuitry': superior posterior parietal cortex, MFG incl. DLPFC & PMC, insula, VLPFC.	Reward valuation: No main effect of group or visit but a significant interaction was observed for all ROIs. Post-hoc analyses: SS choices: HC hungry > HC satiated (all ROIs except left VS); remAN hungry = remAN satiated; HC satiated < remAN satiated. Cognitive control: Significant main effects of group and visit, and significant interaction in left MFG, bilateral insula, right VLPFC and bilateral SPC. Post-hoc analyses: HC hungry < HC satiated (right VMPFC, bilateral insula); HC hungry > HC satiated (left MFG); RemAN satiated > HC satiated (left MFG); RemAN > HC (main effect of group, bilateral MFG); satiated > hungry (main effect of visit, bilateral MFG, left insula, bilateral VLPFC).	Controlled for menstrual cycle. HC were age- and weight-matched. Comorbid psychopathology not controlled for in analyses.

Authors	N	Sample	Reward	Task characteristics	Scanning/analysis procedure	Primary findings: neural correlates of temporal discounting	Other findings/ comments
Studies in Obesity							
Eisenstein et al. (2015)	44	Obese adults (n=24, 4 male) & non-obese (n=20, 4 male). Exclusion: any history of psychiatric diagnosis, including substance abuse/dependence.	Monetary; hypothetical.	Computerised SS immediate & varied; LL fixed ($500). 5 delays (1 week-2 years). 25 binary choices. Outcome: AUC.	PET scanning using a non-displaceable dopamine D2receptor-specific radioligand ([11C]NMB) to quantify striatal D2 receptor binding. ROIs: dorsal striatum (putamen, caudate) and VS (nucleus accumbens). Hierarchical linear regression analyses performed separately for each group (total sample, obese, non-obese).	In the obese group, higher striatal D2 receptor binding related to greater TD. No relationship between TD and striatal D2 receptor binding in the non-obese group. In both groups, striatal D2 receptor binding not related to BMI or percentage body fat.	Age, gender, ethnicity & education included as covariates. Comorbid psychopathology not controlled for in analyses.
Kishinevsky et al. (2012) & Stoeckel et al. (2013)	19	Obese women. Non-smokers, excluded based on presence of ED, current or past substance abuse/addictive disorder, history of ADHD.	Monetary; real (participants randomly chose one task trial & received half of choice at specified time interval).	Computerised SS immediate & varied ($1-73; LL varied ($28-86). Delays 1-116 days. 96 binary choices. 12 sensorimotor control trials ($0 now vs. $0 later). Outcome: k.	A laboratory visit with an offline TD task was used to compute each participants' individual k value. A second laboratory visit 1.3-2.9 years later included only an assessment of weight. Analysis: hard (closer to indifference point) minus easy (more obvious) trials; ROIs (lateral PFC (IFG, MFG, SFG), IPL, SPL, medial PFC (ACC, SFG)) and whole brain analysis.	Kishinevsky et al (2012): harder > easier trials: ROI analysis: IFG, MFG and MPFC (inc. medial SFG and pre-SMA); whole brain analysis: MFG, dACC, middle cingulate, pre-SMA. Negative correlation between weight gain in next 1-3 years and activity in the left IFG, right MFG, right SFG and left IPL activity (ROIs) and left MFG and left dACC (whole brain analysis), during hard (vs easy) trials. Rate of weight gain negatively correlated with activity in the right IFG, right MFG, left IPL (ROIs) and left MFG and left PCC (whole-brain), and positively correlated with right caudate nucleus and IFG activity during hard (compared to easy) trials.	Stoeckel: relationship between TD and activity in the right SFG, left IFG and right PCC remained significant after controlling for age and/education. Controlling for IQ did not alter results. Comorbid psychopathology not controlled for in analyses of both papers.

Authors	N	Sample	Reward	Task characteristics	Scanning/analysis procedure	Primary findings: neural correlates of temporal discounting	Other findings/ comments
						Stoeckel et al (2013): TD correlated with reduced activation in middle & superior frontal gyri, IPL during hard (versus easy) trials (identified by whole-brain analysis). TD negatively correlated with bilateral MFG and right IPL ROIs and trend towards a negative correlation with left IFG ROI. Negative correlations between TD and precuneus, STG and PCC identified by whole-brain analysis.	
Weygandt et al. (2013b)	16	Obese subjects prior to 12-week low-calorie diet (3 male). Excluded based on: affective, anxiety, delusional disorder, borderline personality disorder or substance abuse	Food; hypothetical.	Computerised SS immediate & fixed; LL variable. 22 delay periods (1-120mins). 88 binary choices. Outcome: k.	Performed twice: once offline, once online. Correlations between brain activity and weight loss. ROIs: anterior insula, VS, dorsal striatum, hypothalamus, DLPFC, DMPFC, VMPFC, PCC.	Higher TD associated with poorer dietary success. TD negatively correlated with DLPFC activity (trend) and functional connectivity of the VMPFC with the DLPFC, dorsal striatum and DMPFC. Included motion parameters, cue-reactivity, pre-diet BMI, gender, age and a constant term as covariates of no interest.	Comorbid psychopathology not controlled for in analyses.
Weygandt et al. (2015)	23	Obese subjects on weight modulation programme. Excluded based on: affective, anxiety, delusional disorder, borderline personality disorder or substance abuse	Food; hypothetical.	Computerised SS immediate & fixed; LL variable. 22 delay periods (1-120mins). 88 binary choices. Outcome: k.	2 scanning sessions with assessment of weight: baseline and after 1 year. Analysis: focused on regions that were more active on harder versus easier trials (calculated by median split of reaction time); fMRI contrast: hard-easy trials; ROIs: left and right DLPFC (BA9 & BA46).	Higher TD associated with poorer weight maintenance. Two areas of the left DLPFC (centred in the MFG) and one in the right DLPFC (MFG): hard > easy trials. Activity in right superior frontal gyrus during hard (compared to easy) trials positively associated with degree of weight maintenance. No relationship between change of control-related activity (i.e. DLPFC) and weight maintenance success.	Includes several subjects from Weygandt 2013. TD correlated with both visits (stable marker for impulsivity). Covariates of no-interest: gender, BMI, age, cue-reactivity. Comorbid psychopathology not

Authors	N	Sample	Reward	Task characteristics	Scanning/analysis procedure	Primary findings: neural correlates of temporal discounting	Other findings/ comments
							controlled for in analyses.

AN = anorexia nervosa; HC; remAN; SS = smaller/sooner; LL larger/later; HC = healthy controls; NR = not reported; TD = temporal discounting; T1: time point 1; T2 = time point 2; dACC = dorsal anterior cingulate cortex; DLPFC = dorsolateral prefrontal cortex; ROIs = regions of interest; VS = ventral striatum; VLPFC = ventrolateral prefrontal cortex; MFG = idle frontal gyrus; SPC = superior parietal cortex; PCC = posterior cingulate cortex; ACC = anterior cingulate cortex; PMC = primary motor cortex; PET = Positron emission tomography; ([11C]NMB) = [11C](N-methyl)benperidol; AUC = area under the curve; SFG = superior frontal gyrus; SMA = supplementary motor area; PFC = prefrontal cortex; IFG = inferior frontal gyrus; SPL = superior parietal lobule; IPL = inferior parietal lobule; DMPFC = dorsomedial prefrontal cortex

Table 3. Methodological findings

		Total number of studies	Proportion of studies reporting behavioural group differences in rates of TD*	Number of studies reporting behavioural group differences in TD within each condition			
				AN	BN	BED	Obesity
Reward type	Hypothetical	77% (24/31)	64% (14/22)	-	1	4	9
	Real-life	23% (7/31)	67% (4/6)	1	-	-	3
Reinforces	Monetary	81% (25/31)	62% (15/24)	2	1	4	8
	Food	10% (3/31)	100% (1/1)	-	-	-	1
	Both/Other	10% (3/31)	67% (2/3)	-	-	1	1
Administration of task	Computerised	81% (25/31)	65% (15/23)	2	1	3	9
	Other [a]	19% (6/31)	80% (4/5)	-	-	1	3
Reward framing	Accelerate [b]	64% (20/31)	53% (10/19)	-	-	3	7
	Delay [c]	10% (3/31)	67% (2/3)	-	-	-	2
	Both	26% (8/31)	100% (6/6)	2^	1	1	2
Measurement of TD	AUC	29% (9/31)	44% (4/9)	-	-	2	2
	k	35% (11/31)	62% (5/8)	1	-	1	3
	AUC and *k*	6% (2/31)	0% (0/2)	-	-	-	-
	Other methods [d]	33% (9/31)	100% (9/9)	1	1	1	6
Controlled for in analyses of TD	Age	29% (10/31)	70% (7/10)	2	-	1	4
	Sex	13% (4/31)	50% (2/4)	-	-	-	2
	IQ	16% (5/31)	60% (3/5)	2	-	1	-
	Education	16% (5/31)	60% (3/5)	-	-	1	2
	Income	6% (2/31)	100% (2/2)	-	-	-	2
	Co-morbid psychopathology	13% (4/31)	75% (3/4)		1	1	1

*3 neuroimaging studies did not report behavioural differences; AN: anorexia nervosa; BN: bulimia nervosa; BED: binge eating disorder; [a] e.g. questionnaires, index cards; [b] SS varied, LL fixed; [c] SS fixed, LL varied; [d] e.g. sum of indifference points, mean scores, other slope formulae; ^ one of which reported an effect of framing

Table 4. Calculation and interpretation of temporal discounting outcomes

k value (Richards et al., 1999)	AUC (Myerson et al., 2001)
A hyperbolic function is fitted to the indifference points* for each delay period. A constant, *k*, characterises an individual's rate of discounting and is used to quantify TD rate.	Values are standardised (subjective value and time delay expressed as a proportion of total value/maximum delay) and plot against each other. Summing the trapezoid of each indifference point* generates AUC.
$V = A/(1+kD)$ V = subjective value, A = reward amount, D = delay until receipt and k = slope of TD	$(x2 – x1)*[(y1 + y2)/2]$ x2 & x1 = successive delays, y1 & y2 = subjective values associated with delays
k values range from 0 to 1: larger k values indicate greater TD	AUC values range from 0 to 1: smaller AUC indicate greater rates of TD

*indifference point: the point of subjective equality, i.e. when the larger/later (LL) reward is deemed equal to the smaller/sooner (SS) reward; AUC: area under the curve.